Society Report.

AMERICAN NEUROLOGICAL ASSOCIATION.

ELEVENTH ANNUAL MEETING.

First day, afternoon session.

The American Neurological Association convened at the New York Academy of Medicine, June 17, 1885.

Present—Drs. Ott, Weber, Graeme M. Hammond, Amidon, Rockwell, Seguin, Gray, Birdsall, Gibney, Geo. W. Jacoby, McNutt, and Dana.

The retiring President, Dr. Isaac Ott, of Easton, Pa., called the Association to order at 3 P.M., and after the following remarks introduced the Vice-President-Elect, Dr. Leonard Weber, of New York.

THE PRESIDENT'S ADDRESS.

Gentlemen: It devolves upon me to make a short address before retiring from the chair. In looking back over the past year I can, without hesitation, state that neurological science has been advancing with rapid strides, and that the physiology of the nervous system has not been at a standstill. If I may be permitted to refer to a subject upon which I have been working, I feel safe in forecasting that in the near future the relation of the nervous system to the temperature of the body will be an object of much important research. This problem is of considerable interest to the neurologist, and of much more value to the general practitioner. The researches of various observers lead up to the conclusion that the temperature of the body, like the heart, is under the control of the nervous system. Admitting this statement to be true, the next inquiry is the location of these centres. Tscheschichin, nearly twenty years ago, believed a moderating centre to be in the brain. Eulenburg discovered a centre at the sulcus cruciatus which can cause a great rise in temperature, but lately he believes the increment of heat to be due to vaso-motor disturbance. In the JOURNAL OF NERVOUS AND MENTAL DISEASE for 1884, I published a paper showing that in the vicinity of the corpora striata were centres having a relation to the temperature of the body. Lately, Messrs. Aronsohn and Sachs have arrived at the conclusion that in the cerebrum are similar centres, whose position corresponds to the vicinity of the corpora striata. During the past few months I have attempted to more definitely map out the centres, and find them located in the anterior inner part of the optic thalami. Now the rise of temperature is due either to decreased dissipation or increased production. I made use of d'Arsonval's calorimeter, surrounding it with felt and feathers. Voit's respiration-apparatus served to note the quantity of air by its large meter and at the same time was used as an aspirator. I have found these centres to be concerned in heat production, thus elevating the temperature, and not generating it by lessened dissipation. These centres are then veritable heat çentres, and not due to vaso-motor deflections, for in the latter case there would be lessened dissipation of heat. The cortex of the brain has not any thing to do with this rise of heat, as you can obtain the increase no matter at what part of the surface of the brain the needle enters. Further, removal of different parts of the cortex causes only a temporary rise, whilst the rise in the centres concerned lasts two or three days, and often amounts to about 4½° F.

The next order of business was the reading of the minutes of the last annual meeting. Dr. AMIDON moved that, as they had been published, the reading of the minutes be dispensed with. Carried.

The Treasurer made his report, which was accepted.

The Council reported, recommending the acceptance of the resignation of Dr. Mason as active member, and that he be made an associate member. Dr. Mason's resignation was accepted, and he was then elected an associate member.

The Council recommended the election to active membership of Dr. Philip Zenner, of Cincinnati, Ohio, and of Dr. M. Allen Starr, of New York. Both gentlemen were elected.

The Secretary read communications from Drs. J. S. Jewell and H. Gradle, of Chicago; S. F. Danillo, of St. Petersburgh; H. D. Schmidt, of New Orleans; C. K. Mills and J. T. Eskridge, of Philadelphia; Augustus Forel, of Zurich; and Roberts Bartholow, of Philadelphia. On motion, the communications were received.

NOMINATION OF OFFICERS.

For President, Dr. AMIDON nominated Dr. C. K. Mills, of Philadelphia.

On motion, the nominations for President were closed.

For Vice-President, Dr. V. P. Gibney, of New York, was nominated.

On motion, the nominations for Vice-President were closed.

For Secretary and Treasurer Dr. BIRDSALL nominated Dr. R. W. Amidon, of New York.

On motion, the nominations for Secretary and Treasurer were closed.

For Councillors, Dr. AMIDON nominated Dr. George W. Jacoby, of New York, and Dr. GIBNEY nominated Dr. E. C. Seguin, of New York.

On motion, the nominations for Councillors were closed.

On motion, the Secretary was instructed to cast an affirmative ballot for all the candidates nominated for office for the year 1886. The Secretary so cast his vote, and the Chairman declared the gentlemen elected.

The two following proposed amendments to the constitution and by-laws were then called up:

To amend Article VI. of the constitution, by striking out the words "third Wednesday in June," and inserting the words "first Wednesday in May." Proposed by Dr. W. A. HAMMOND.

All business not of a scientific nature shall be transacted in executive session. The executive session shall be held after each session. Proposed by Dr. E. C. SPITZKA.

After a discussion of the first proposed, amendment by Drs. AMIDON, SEGUIN, GRAY, GIBNEY, and BIRDSALL, the amendment was laid on the table, and Dr. SEGUIN substituted the following:

Art. VI. The annual meeting of the Association shall be held on any two or three days in the month of July in each year as may be chosen by the Council. The Council shall also designate the place of meeting.

Dr. E. C. SEGUIN then moved the adoption of the second amendment.

After a discussion by Drs. GIBNEY, SEGUIN, GRAY, JACOBY, and BIRDSALL, the amendment was carried.

The Association then proceeded with its scientific work.

Dr. A. D. ROCKWELL, of New York, reported "A Case of Chronic Myelitis; Recovery."

The complete recovery of any case of chronic, supervening on acute, myelitis, in which all the most desperate features of that disease have been illustrated, is so rare an occurrence that, when such a result is claimed, the case should be subjected to the closest scrutiny. With this object in view I offer the following for your consideration and critical judgment:

Miss B., aged sixteen, was referred to me by the family physician, Dr. J. O. Farrington, of this city, September 22, 1884.

Three months previously she went on a Sunday-school excursion to the sea-shore, and all through the day experienced more or less physical depression, and, instead of actively engaging in exercise, she sat much of the time on rocks, near the water, which may have been damp from the tide. Upon returning from the picnic she felt cold, and was so bent over that she could walk only with difficulty. During the night and for several days she suffered acutely, and within a week the lower limbs became completely paralyzed, as well as the sphincters of both rectum and bladder. She suffered from an aggravated form of constipation, and from incontinence of fæces after the aperient that was regularly called for. Incontinence of urine was a constant symptom, supplemented by a severe attack of cystitis, and accompanied by acute pain.

After six weeks of suffering a bed-sore made its appearance in the region of the last lumbar vertebra. It resisted all treatment, and rapidly enlarged until it was about two inches in diameter.

For two months she lived almost entirely on milk and champagne. When I saw the patient she was emaciated to the last degree. Electro-muscular irritability to both currents was entirely abolished, the sphincters were paralyzed, the legs were contracted beyond the power of forcible extension, and the bed-sore was large, with a tendency to increase. The limbs were sensitive to the touch, with the paradoxical symptom of diminished sensibility when tested with the æsthesiometer. A marked perversion of sensibility was evidenced by the exceedingly disagreeable numb feeling excited by the induced current.

The faradic current had been used for several weeks previously, but, as was to be expected, without any effect whatever. I immediately began galvanizing the legs and the lower portion of the spine, using a current strength of about thirty volts, and repeating the applications every other day. Three weeks of persistent effort failed to elicit any response in the way of muscular contractions, and the only evidence that the treatment might possibly be doing good service was the improved condition of the bed-sore. At the end of the fourth week very faint muscular contractions were observed, and within another week they had so increased in vigor as to render it possible to interpret their character. They were markedly the reactions of degeneration. From this time onward the patient steadily improved. The electrical reactions became quite normal, and within a month the bed-sore had entirely healed. The patient regained power over the bladder more rapidly than over the bowels; but within two months she had complete control over both fæces and urine. She was treated some four months, and at the end of that time was discharged as cured, with the exception of slight weakness in the flexor muscles of the right leg. This weakness still remains, indicating a possible irreparable damage to certain nerve-cells.

There can be but little doubt that this case was in the beginning one of acute inflammation of the cord. What the exact pathological condition was in its secondary and chronic stage may be a question involving a greater difference of opinion.

The tendency probably was either to degenerative softening or atrophy of the ganglion cells, and, to my mind, it was the second rather than the first pathological change that prevailed in this case; and that the whole transverse area of the cord was involved was evidenced by the complete abolition of both afferent and efferent nerve function. The practical value of this case, so far as relates to the pathological side, is in its distinct negation rather than in any affirmation. It is self-evident that, however much structural change had taken place in the way of atrophy of nerve-cells, or induration or overgrowth of connective tissue, the nutrition of these nerve-cells had not been sufficiently impaired to be beyond the influence of the reparative process.

In its therapeutic aspect the lesson taught cannot be too strongly emphasized.

The father, in the face of a completely discouraging prognosis, insisted that unceasing effort should be made, and subsequently his triumphant but somewhat reproachful thought was: "What would have become of my child if I had been guided by the professional opinion so confidently expressed?"

Remarks on Dr. Rockwell's Communication.

Dr E. C. SEGUIN: I am very much interested in the case. It reminds me somewhat of a number of cases I have seen in which the typical picture of poliomyelitis was obscured by the occurrence of weakness of the sphincters and slight dulness of sensibility. I believe that was all there was here. There was no absolute anæsthesia. It seems to me this case would come under the category of cases of modified poliomyelitis, with some extension into the central gray matter. It certainly cannot be brought forward as an example of cure of myelitis transversa. It leaves our unfavorable prognosis of that disease, from which I have never seen complete recovery, unchanged. I saw a case of poliomyelitis with Dr. Delafield about six years ago, in which the symptoms were nearly as severe as in Dr. Rockwell's case. There was retention of the urine, with paresis of the arms and legs, hyperæsthesia and very severe pains extending along the principal nerves. It seemed to be a case of diffuse myelitis, most marked, however, in the anterior cornua. That patient recovered entirely within two years, and is now doing the duties of an active citizen. Dr. Rockwell's case is exceedingly interesting, and is particularly instructive with reference to treatment. The time employed in the treatment by galvanism of course allowed of a chance of spontaneous restoration; but I have no doubt that galvanism did something toward expediting the cure by improving the nutrition of the affected parts.

Dr. ROCKWELL: In the case seen with Dr. Delafield, were the sphincters affected?

Dr. SEGUIN: The bladder was paralyzed, if I remember right.

Dr. ROCKWELL: In my case the sphincters were paralyzed for many weeks, there being complete inability to prevent the passage of the urine and fæces. I have seen quite a number of cases of poliomyelitis anterior. This is not a typical case of poliomyelitis anterior; it is a typical case of myelitis, with the exception that there was no complete anæsthesia.

Dr. M. A. STARR asked Dr. Rockwell whether the treatment of the case was solely by galvanism, or whether other means were used which might possibly have had some effect in bringing about recovery. Furthermore, he would like to know how galvanism was applied; whether by interruptions to the muscles only, or whether the constant current was also applied to the cord.

Dr. ROCKWELL: No other remedies were employed except those resorted to in the beginning, and supporting measures, as cod-liver oil, etc.; and above all no other remedies directed to the disease itself. The current was directed to the muscles and also to the spinal cord, and I applied electricity to the spinal cord by means of very sharp and strong interruptions, as well as by means of the constant current, I thought that as there was no irritability at all it might be well to use the strong interrupted current as well as the steady current, in hopes that some improvement in nutrition might take place.

Dr. W. R. BIRDSALL: I would ask Dr. Rockwell whether all of the muscular groups were affected alike; whether there was absolute paralysis of all of the muscular groups in the lower extremities. Also as to the limitations of the sensory symptoms, which he said were slight. Also whether any observations were made with regard to the skin reflexes. I believe nothing was stated with regard to the tendon reflexes. It seems to me those are important points in enabling us to determine whether there was poliomyelitis anterior or a transverse myelitis. I think we may have conditions in which a central myelitis of a transverse character may occur, possibly with slight involvement of the lateral columns, also of the sensory tracts in the posterior columns, making certain cases resemble strongly an ordinary case of myelitis of the anterior horns. I believe these cases are quite rare. It does not appear to me from the case, as stated, that we can regard it as one of anterior myelitis with involvement of the central tracts and lateral columns, especially as the sensory disturbances were of a slight character.

Dr. ROCKWELL: All the groups of the muscles of the leg were affected alike. I thoroughly tested all the muscles of the leg, and there was absolute loss of contractility in every muscle and fibre. With regard to the sensory symptoms, those were observed only in the toes. With regard to the patellar reflexes, I omitted to state that they were absent in the beginning, but they have returned. With regard to ankle clonus, I remember there was marked agitation of the foot.

Dr. V. P. GIBNEY: Is it possible to have absence of patellar reflex and marked ankle clonus? It is my impression that where there is absence of patellar tendon reflex there is also absence of ankle clonus.

Dr. ROCKWELL: I did not test the case for ankle clonus. I merely remarked that there was considerable motion of the foot.

Dr. GRAY, of Brooklyn: It depends upon what you call chronic myelitis as to whether you are going to regard recovery from it as possible or not. To take a case which appeared to be absolutely hopeless, which had not responded to treatment for months after the natural time for recovery had passed, and then to have it suddenly under a certain method of treatment recover, that would be quite a therapeutic discovery. But we do not know, after all, very much about myelitis. We can diagnosticate myelitis of the anterior horns; we can diagnosticate a myelitis of the columns of Burdach, a locomotor ataxia, or of the lateral pyramidal columns; but autopsies and experimental investigations have shown us that there are probably many other columns of the spinal cord which are entirely distinct from one another in their functions, but whose symptomatology in disease is unknown. But from what we know of the symptoms arising from disease of the anterior horns, of the columns of Burdach, and of the pyramidal columns, it is very likely that the unknown columns also give rise to distinct symptoms in disease. When we come to speak of myelitis proper, we mean to say only that the patient has some form of myelitis that is not disease of the anterior cornua, or of the lateral pyramidal columns, or of the columns of Burdach, or of columns which are known in disease to give rise to definite symptoms; yet in that generic term, myelitis, may be embraced half a dozen totally distinct maladies. I have seen a good many cases which have lasted a long time seemingly, yet have finally made a perfect recovery. I have in mind one case, which Dr. Seguin will recall, that of a California gentleman, in which it required a year for recovery to take place. The first symptom which alarmed the patient occurred while he was in Virginia City. Within a week he reached San Francisco, and his symptoms getting worse, he started for New York. By the time he had reached the Isthmus of Panama he was completely paralyzed in the lower extremities. It was upward of a year before he recovered, and more than three months before there was any retrocession in his symptoms. I have seen several cases like that. So that the fact of recovery from rather chronic myelitis does not occur quite so seldom as Dr. Rockwell's experience would seem to show. I have noticed once or twice cases apparently quiescent for perhaps months, nothing seeming to be of any benefit, when quickly, after beginning the use of electricity, either the faradic or galvanic current, improvement would take place. I recall one case of typical myelitis of the anterior horn which had been in a quiescent condition for several years. The patient came to my clinic, and I gave a very unfavorable prognosis; and, in order to keep the patient coming to the clinic as the subject of a lecture, I administered a powerful faradic current, and to my great surprise the improvement which took place in his condition could be estimated at about fifty per cent. I have seen the same thing occur in another case.

Dr. ROCKWELL: In closing the discussion I have nothing to say except that my case was a very gratifying one in its results. We would like to know what had been the exact pathological condition, but of course that question cannot be decided positively. The case was especially interesting to all of us, as showing that there was very probably a direct relation between the therapeutics employed and the recovery. Whether in time recovery would have taken place spontaneously is a question, but it seems to me exceedingly improbable, from the fact that so many weeks had gone after the acute symptoms had passed without any improvement being manifest, there being, on the contrary, an apparent progression of the disease. I think that more can be accomplished in the treatment of certain of these cases of myelitis by persistent effort than has been supposed to be possible.

Dr. M. A. STARR then read a paper entitled, "Recent Methods of Staining" (published in full in the JOURNAL OF NERVOUS AND MENTAL DISEASE, New Series, vol. x., p. 143, 1885).

Remarks on Dr. Starr's Communication.

Dr. E. C. SEGUIN: Has Weigert's method been used for large or transverse sections of the brain?

Dr. STARR: I have some transverse sections of the dog's brain stained after this method, and the result is very satisfactory. Not expecting to read my paper to-day, I did not bring them with me.

Dr. SEGUIN: With regard to Sankey's method and my modification of it, I may say that my attention was directed to its use while making sections of a brain in Zürich, in the summer of 1883. The history of this brain was not fully known, and it was uncertain whether it had not for some time lain in alcohol. At any rate it did not respond well to carmine, but, as usual when a specimen has been in alcohol for any length of time, the results were not bright or distinct. I then thought of other methods, and came across that of Sankey. But by this method I found large sections intractable; having to be cut very thin, like a veil, they were difficult to manipulate, and became invisible when immersed in the solution. I therefore reduced the strength of the solution to two and to four thousand. Two thousand gave good results after six or ten hours. Three thousand enabled me to leave the specimen in the fluid overnight, which was an advantage. By this method, adopting a certain routine, one can prepare a large number of sections in the course of a few weeks. The only advantage I claimed for the method was for lens or naked-eye demonstrations. I also examined these sections with the microscope, and the ganglion cells came out very well, although possibly with the fault which Dr. Starr mentioned. I have also tried the method in preparing pathological specimens, and the reaction is very satisfactory.

The Society then adjourned, to meet at 8:30 P.M.

First day, evening session.

The Association was called to order at 8:30 P.M. by the Vice-President, Dr. WEBER.

Present—Drs. Weber, G. M. Hammond, Starr, Seguin, Dana, McNutt, Amidon, and Jacoby.

The minutes of the afternoon session were read and adopted.

Dr. GEORGE W. JACOBY then read a paper " On the Use of Osmic Acid in Peripheral Neuralgias."

Dr. Jacoby, after referring to the cases published by Neuber, Eulenburg, Wölfler, and Merces, said that particularly the results attained by the two latter were such good ones that it became imperative to corroborate them, or, if possible, to refute them. For this purpose he publishes a set of cases in which he used the remedy.

The manner in which he treated his cases was as follows:

He made use of a one-per-cent. solution of osmic acid in water, and of this 0.50 to 1.00, equal to 0.005 or 0.010 of the acid, was injected. In a few cases less was used, but very rarely. Dr. Jacoby said, the preparation was that known as osmium tetroxide, OsO_4, commonly known in histological research as osmic acid. The preparation known as hyperosmic acid, which was used by Neuber and others, is probably the same. According to the best treatises on chemistry, such a combination does not exist, as no mention is made of it. The solution, when exposed to the light, rapidly becomes decomposed, turning dark, and ultimately quite black. Eulenburg says that it can, nevertheless, be used in this condition, but his (Jacoby's) experience has convinced him of the contrary. He considers it entirely inactive when in this state. For this reason it should be dispensed only in a dark bottle, and a small quantity only ordered at a time. He never prescribed more than 20.00 of the solution, this quantity serving about forty injections. The injections were always made as near as possible to the point of severest pain, and into the connective tissue surrounding the affected nerve. The pain caused by the injection is in many cases exceedingly severe, producing a stinging, burning sensation, which generally follows the course of the nerve. This pain, however, rarely lasts long, generally subsiding after a few seconds. Occasionally, also, local changes are produced, such as swelling and puffiness over the seat of puncture, and in many cases he has seen a hard circumscribed thickening of the skin and connective tissue produced in consequence of repeated injections, which only disappeared after days. There is no discoloration produced by the acid, except perhaps a single black spot where the needle was introduced. The action is a purely local one, no constitutional symptoms whatever being produced.

If various branches of a nerve are affected, it is necessary to inject over each painful branch. These cases will, however, be found to be mostly intractable ones. Certainly the results are better in those cases in which the pain is localized over a single branch. The nerves of the skin do not appear to be at all affected by the injections. and no anæsthetic action was discernible in any of his cases.

Three cases were then read in detail; two for the purpose of showing the general plan of treatment, and the third for the purpose of showing a possible injurious action of the acid. This case was as follows:

Female, æt. forty five. Seen in consultation April 12, 1884. Left cervico-brachial neuralgia. Principal seat of pain over the radial in the arm. The pain had existed for two years, and was probably the result of a periarthritis of the shoulder. Every remedy that had been suggested had been tried, but without result. I recommended a trial of the osmic-acid injection, and made one myself over the musculo-spiral nerve. I heard nothing more of the case for a fortnight, the attending physician having promised to continue the suggested plan of treatment. After the lapse of that period of time, on April 26th, the physician called upon me and told me that the case had progressed favorably until three days prior to his visit. The pain had gradually decreased, and prospects of an entire recovery were fair. At the time mentioned he made the sixth injection in the usual place. The patient complained at once of intense pain radiating downward into the thumb and forefinger. The pain at previous injections had been merely nominal. A few hours after that she called again and said that she had a feeling of numbness in the first fingers and along the outer border of the arm. The following morning the physician called upon her and found a well-marked classic radial paralysis. The patient had during the night slept with her arm thrown backward and under her head, and the doctor concluded that he had to deal with a common case of radial paralysis due to pressure. To me the case was not so clear. The sudden severe pain along the course of the radial after the injection, the advent of anæsthesia soon after, inclined me to the view that the paralysis was due to the injection itself.

Dr. Jacoby's cases, when analyzed, show us of 18 cases: 8 cures, 2 improved, and 8 unaffected. Of the 8 cured cases, 5 were cases of sciatica. All the cured cases, including the sciaticas, were old cases. Of the unimproved cases, 8 in number, 3 were fresh cases and two were old. The case marked 8 cannot be counted, and of 12 and 14 no note is made.

At any rate it would seem from these cases that the sciatic nerve is the one which is most impressionable to the action of this remedy, and that old inveterate cases present a greater chance of success than fresh ones. This is contrary to Eulenburg's experience, and in the present set of cases may be misleading. The remedy was used only exceptionally upon fresh cases, in the majority of instances it being used only as an *ultimum refugium.*

The conclusions then drawn were:

1. We have in osmic acid a remedy which is of service in the treatment of certain cases of peripheral neuralgia and in some cases where every other remedy has failed.

2. Osmic acid is not an antineuralgic; its action is very localized, and it frequently fails where other remedies succeed.

3. Its employment is, in most cases, very painful, and not altogether free from danger.

4. In view of Case 8, it is dangerous to implicate a motor nerve in the injection.

Remarks on Dr. Jacoby's Paper.

Dr. SACHS, by invitation: As Dr. Jacoby has referred to the fact that I have had opportunity to observe some of the results obtained by the use of this remedy, I would say that the experience which I have had with osmic acid was only in part satisfactory. Unfortunately I did not hear all of Dr. Jacoby's paper, and am not informed as to the exact conclusions at which he arrived. I am compelled to acknowledge that the remedy is unreliable in many cases. The only good results which I saw obtained from it were in cases of old standing, more particularly in cases of sciatica. I can recall several cases of sciatica which were under my care while I was in charge of Prof. Eulenburg's clinic in Berlin, and which did not yield to the usual form of electrical treatment. In these cases marked improvement was noted after repeated injections of osmic acid over the seat of the trouble. I believe that but one third of the cases reported by Eulenburg himself were treated successfully by this method. In my opinion the drug is worthy of a trial, particularly where other remedies have been employed without success.

Dr. E. C. SEGUIN: I have not had opportunity to use osmic acid, but I have had some experience with treatment upon the same general principle. The method of treating neuralgias by the deep hypodermic injection of nitrate of silver and other irritating remedies was brought forward by Luton as far back as 1863. In 1864 I think it was that I treated one case of rebellious sciatica by that method, and in the Strangers' Hospital, a temporary institution in existence here about the time of the collapse of the Ring in 1873–4, I treated another case. Both cases were successful, and in both the neuralgia had existed for ten or thirteen years. The injections were made deeply and gave rise to abscesses. The solution employed was that recommended by Luton. There must have been an inflammatory reaction set up about the nerve. In the case treated in the Strangers' Hospital the small abscess healed quickly. But in a case treated, I believe, on David's Island in 1864, the abscess became fistulous, and remained for many months a source of trouble. Luton relates many cases of neuralgia, particularly old ones, treated successfully by this method. It acts by producing counter-irritation close to the nerve. Hypodermics of quinine, I suppose, act on that principle. I published in the *New York Medical Journal*, August, 1867, cases of malarious neuralgia treated by hypodermic injections of quinine *loco dolente;* and I then suggested that the effects were probably largely due to the local effects of the irritating injection, as well as to the constitutional effects of the quinine. It seems to me that the general method, including a variety of remedies which may be employed for the injection, offers a large scope for successful practice. We may be able to find a substance which is not so liable to be irritating or to give rise to local trouble as osmic acid or nitrate of silver. Perhaps iodine or ether would do in many cases.

A word with regard to numbness and paralysis following deep injections over a nerve. I know of two such cases, one occurring in my own practice and the other in that of a friend. Five years ago I had a case of trigeminal neuralgia in which I made two hypodermic injections of chloroform on two different days near the infra-orbital foramen, according to the plan advocated by Dr. Bartholow. There was some relief of pain, but after the second injection there was very complete anæsthesia of the upper lip, which continued as long as I had the patient under observation, which was for several months. The other case was identical with this one. The physician gave, at my suggestion, I believe, a hypodermic injection of chloroform in the cheek. The drug entered too close to the nerve and produced anæsthesia of the upper lip. I have never seen a motor nerve affected by this treatment.

Dr. JACOBY: The remarks made by Dr. Sachs coincide pretty well with the conclusions I have drawn in my paper. I believe my successes were a little over a third. But the difference between Eulenburg's experience and my own in the causation of pain by the injection was notable. In the majority of my cases the pain produced by the injection was very severe, and may have been due to the preparation of the acid; but beyond the case of radial paralysis I have seen no bad effects from it. No bad effects followed the injection in the other fifty cases, or more, referred to. As to whether the acid acts only as a counter-irritant, I do not think we are able yet to say. The fact of discoloration of living tissue in Fraenkel's experiments might lead us to suppose that the drug interfered in some unknown way with nerve action. I should not recommend the use of osmic acid in every case of neuralgia, but it is well to know of the remedy and to use it when occasion demands.

Dr. SEGUIN: Does Eulenburg use a discolored solution? I should think that inert, from histological experience

Dr. JACOBY: Eulenburg uses a fresh solution, but he makes the remark that when it becomes discolored its action is not interfered with; it acts just as well as when fresh. But I find it different. The first cases I treated with an old solution and I obtained no results. Then I began a new set of cases with a fresh solution.

Dr. SEGUIN further remarked that Ranvier had employed hypodermic injections of osmic acid for the purpose of demonstrating the normal nerve-fibres, and also for obtaining specimens of fat-cells. It was a very successful method.

The meeting then adjourned.

Second day, afternoon session.

The Society was called to order by the President, Dr. BURT G. WILDER, at 2:30 P.M.

Present—Drs. Wilder, Weber, Seguin, G. M. Hammond, Gibney, Amidon, Starr, Gray, Jacoby, Birdsall, Dana, and Rockwell.

The minutes of the previous meeting were read and approved.

Dr. V. P. GIBNEY then reported two cases of spina bifida cured: the first by antiseptic incision, followed within a fortnight by chronic hydrocephalus, frequent aspirations of the head, and death from hydrocephalus; the second, a case cured by injections of the iodo-glycerine solution, according to the method of Morton, of Glasgow. The cases were presented for the purpose of directing attention to the radical cure of spina bifida.

CASE I.—A male child seven days of age came under his observation in July, 1884. The tumor was situated over the sacrum, and was well covered with normal skin except in its lower portion, where was seen a small indolent ulcer about half an inch in diameter, covered with a thin whitish crust. There was no hydrocephalus, nor any deformity of the limbs, and to all appearances the functions were normal. The sac was translucent, and no traces of nerve tissue could be seen in it. There was no history of hereditary influence. Two weeks later the tumor had perceptibly enlarged, and Dr. Gibney aspirated it once or twice, and injected the iodo-glycerine solution. He introduced the small needle of the aspirator and removed about one drachm of purulent fluid. This surprised him so much as to render his diagnosis doubtful, but further examination and consultation confirmed the original diagnosis of spina bifida, and the change from serous to purulent fluid was explained by the contiguity of the ulcer with the sac. Two days later the limbs were subjected to the faradic test, and the reactions in all the muscles were preserved. The patellar reflexes were much exaggerated, while there was also a marked ankle clonus. The rectal sphincter was paralyzed. The temperature was 100.2° F. Four days later, with strict antiseptic precautions, he opened the tumor with a bistoury and gave exit to a small quantity of pus, and dressed the wound antiseptically. Two days later the reflexes were less marked, and the general condition of the patient was improved. Four days subsequently there was prolapsus of the rectum and a profuse furuncular eruption over the lower half of the body posteriorly. The wound had healed and the tumor had appreciably diminished in size. Within a week the limbs were acting normally, but the prolapsus of the rectum continued.

Twenty days after the operation, the child then being six weeks old, there was unmistakable hydrocephalus, and from this time forward the spina bifida gave no trouble, and the solidity of the tumor convinced him that a cure had been wrought.

For the hydrocephalus he began a series of aspirations, and by the first removed two ounces of clear serum, having introduced the needle into the anterior fontanelle, to the right of the longitudinal sinus. No untoward symptoms followed, and the aspiration was repeated nine days afterward, when only one ounce was removed. The head continued to enlarge despite the two aspirations. About one month subsequently he aspirated again and removed three ounces of fluid. The subsequent history of the case consisted simply of a repetition of the operation with loss of sight, stupor, and finally death at the end of three months after the first aspiration.

At the autopsy Dr. Gibney had the assistance of Drs. Holt and Amidon. Section through the sacral tumor was quite difficult, fully one inch of dense cicatricial tissue being penetrated before the bony parts were reached. At the bottom of this mass was a small sac, the capsule of which was about ten millimetres. It contained no fluid whatever, but it communicated with the vertebral canal by a pin-point aperture through which a straw was inserted between the dura and the arachnoid to the point where the cauda equina begins. The laminæ of the sacral vertebræ were wanting, and the space was filled with dense fibrous tissue extending up into the canal.

On microscopic examination the dura was found thickened, most markedly posteriorly, and gradually getting thinner until the point of giving off the posterior roots was reached, when it assumed about its normal thickness ; over the anterior and lateral portions of the cord it was normal. Its greatest thickness, two millimetres, was in the lumbar region ; in the cervical region one millimetre thick. The inner surface was coated with a layer of lymph and lymphoid cells of varying thickness. The pia appeared everywhere thickened and infiltrated externally with some products of inflammation, and it seemed abnormally loose in its attachments to the cord. Dr. Amidon made this examination.

The tissue of the cord was apparently normal, unless it were unusally friable in the posterior column.

It was Dr. Gibney's opinion that the child had meningitis, chiefly confined to the posterior surface of the cord, and that the exudation when fresh might have, in places, produced a paraplegia from pressure.

The anterior fontanelles were much depressed ; the sutures were all widely separated. There was only a very small quantity of fluid within the cranial cavity ; the hemispheres were quite soft, and fluctuation was very marked. On puncturing the cortex four pints of serum escaped, nearly the whole quantity coming from the lateral ventricles. The remaining cortex was about one line in thickness. The ependyma was quite granular, and on the right side presented four or five minute points, unquestionably wounds from the aspirator needle. There was no evidence of recent inflammation of the membranes of the brain. The cerebellum and medulla presented no changes microscopically.

The existence of chronic hydrocephalus and spina bifida in the same subject is not uncommon, and indeed one would infer that in this particular case the hydrocephalus was present in fœtal life, and by pressure of the fluid on the medulla contributed to the arrest of development of the vertebral arches at the end of the spinal column. Dr. Gibney was quite positive, however, that during the first four weeks of life there were no appreciable signs of hydrocephalus.

It was with some misgiving that he operated on the spina bifida in this case. It seemed, however, that if pus were contained within in the sac the child was doomed, and he could see no good reason for not operating. In searching the literature of the subject he found that he had good authority for such a procedure. Dr. Gibney then quoted cases reported by Mr. Robert Howsen and Mr. Mayo Robson. Considering the anatomy of spina bifida it would seem that excision of the sac is limited to a comparatively small number of cases. Mr. R. W. Parker, member of the committee to investigate this subject, says that in over one hundred specimens examined in the museums of the London hospitals, ninety-five per cent. contained some part of the cord within the sac. According to Professor Humphrey, of the University of Cambridge, it is pretty well demonstrated that in the large percentage of the cases, not only the cord but the spina bifida, are contained within the wall of the sac.

Case 2.—A female child, three weeks of age ; spina bifida in the sacral region ; the child well developed and otherwise free from deformity ; tumor sessile, and about the size of a small tomato, smooth, and covered with reddish-blue membrane. Dr. Gibney treated the tumor by compression for nearly two weeks, but then aspirated it, removing a drachm and a half of clear serum. He repeated the operation six days afterward, removing only about half a drachm of serum. Six days subsequently, with the assistance of Dr. L. Emmet Holt, he withdrew two ounces of serum by means of the aspirator, and injected with the hypodermic syringe a half drachm of the following solution : ℞ Iodin. resub., gr. x. ; potass. iodid., ʒ ss. ; glycerina, ℥ i. The operation was performed with the child in the dorsal decubitus, the puncture sealed with collodion and cotton, a smooth compress applied, and the child kept in this position for forty-eight hours. No untoward symptom developed. Ten days afterward the tumor was one half its original size, and the tissues were denser, and there was no translucency. Ten days subsequently the cure was complete, and the case was dismissed. Four months afterward there was only a hard, flattened, cicatricial mass where the former tumor had existed. Three days subsequently the child died of cholera infantum, and no autopsy could be obtained.

One would think that the injection of the sac would be dangerous, and indeed speedy death had in some instances followed the operation. Remembering, however, the anatomy of spina bifida, so well demonstrated by Prof. Humphrey, it is easily seen how harmless the fluid may be. He states, when speaking of this treatment: "You will perceive that the separation of the cavity of the sac from the cavity of the arachnoid in most instances is some security against the direct passage of the injected fluid into the latter cavity, and that the advantage of Morton's fluid as an injection probably depends upon the addition of glycerine to the iodine and iodide of potassium, lessening the liability to the diffusion of those irritating substances through the delicate lining of the sac into the surrounding subarachnoid tissue."

Dr. Gibney concluded his paper by saying that in view of the anatomy of spina bifida the plan advocated by Mr. Morton, of Glasgow, seemed to be altogether safer than excision, and also more scientific. With good surgical judgment to guide one, and a knowledge of the parts implicated, the majority of cases of spina bifida ought to be amenable to treatment.

Remarks on Dr. Gibney's Communication.

The PRESIDENT asked those who had had opportunity to examine such cases whether they had noticed dilatation of the aqueductus Sylvii or Iter. He thought he had noticed in reports of hydrocephalic cases that this particular passage seemed to resist dilatation.

Dr. L. C. GRAY read a paper "On the Use of Strychnia in Nervous Affection." He referred to a paper read by Dr. Jewell, of Chicago, before the Association in 1879, in which it was claimed that large doses of strychnia, even as much as $\frac{1}{10}$ grain, could be given with gread advantage in myelitis. Dr. Gray had given it in a number of cases, with varying results, but had never found it safe to go beyond $\frac{1}{25}$ of a grain. In two cases, cumulative symptoms appeared, and in one the patient came near dying. In progressive muscular atrophy the drug could be given steadily for a long time, in one case over a year, with the effect of a valuable stimulant, but not effecting any real improvement. In neurasthenia it acted capriciously—sometimes admirably, sometimes badly. In myelitis of the central type it acted differently in different forms and at different stages. Thus, a chronic myelitis of traumatic origin has greatly improved, as has also another of idiopathic origin ; a myelitis (central), with early implication of the lateral columns, was the one that came near dying ; whilst some cases of poliomyelitis anterior in the early stage were badly affected by it.

Remarks on Dr. Gray's Communication.

Dr. C. L. DANA.—I have had some rather interesting experience in the use of strychnia in some of the functional diseases of the nervous system lately ; I have had under observation lately three patients to whom I have been giving large doses of strychnia. One is a case of depressive form of spinal irritation. The patient is about twenty-five years old, and has been obliged to abandon his business on account of physical and mental inability to continue it ; his back gave out, and his head gave out, and he complained of a long train of symptoms seen in such cases. I gave him strychnia in solution, so that fifteen minims equalled a fifteenth of a grain of the drug. He began with that amount, and increased the dose until he took, in twenty-four hours, over a grain of strychnia in three doses (one of a hundred drops, one of sixty, and the third of thirty). Then he began to feel some effects of the drug—incoördination and palpitation,—and he had to lie down. But those symptoms soon passed off. I then reduced the dose, and he took about sixty drops three times a day for four or five weeks. He says he had never received as much benefit from any other medicine. He has on the average been taking over three quarters of a grain of strychnia daily for over four weeks, with great benefit. There might be some question whether the drug was pure, but I asked the druggist about it, and he assured me that it was, and I tried the drug and found that a dose of a fifteenth of a grain would affect me, though it is an amount which I can generally take without inconvenience.

In the two other cases I ran the dose up until the patients took from an eighth to a sixth of a grain three times a day, and I can also recall other cases in which I have given about that amount. I have found that there are a good many cases of what formerly would have been called spinal anæmia or a depressed nervous condition, in which these large doses can be borne, the patients claiming greater benefit than from any other drug. I think, therefore, that our ideas upon the subject of dosage of strychnia ought to be somewhat modified. I appreciate that it is a dangerous drug to deal with. I know that Nothnagel and some others claim that it is cumulative in its effects. If given in pill form it would certainly be dangerous to give it in increasing doses. But I do not think there is any danger if the patient be an intelligent man and the doctor cautions him. Certainly I have never seen the cumulative or explosive effect of strychnia in any of the cases in which I have administered it.

The physiological effects, or the symptoms which I have noticed, have in some cases been interesting. In some cases I have seen large doses take away that pain and weariness in the back and back of the neck which are so early and constant symptoms of exhaustion. In other cases the drug has imparted tone to the sexual system. It produced remarkable effects in relieving insomnia in one patient to whom I gave it. Bromides and other remedies had failed.

Dr. E. C. SEGUIN: It seems to me we have not made very much advance upon our former knowledge of the dosage of strychnia. I was taught a good many years ago by Brown-Séquard the same ideas I have heard expressed this afternoon. That is to say, in functional affections, more particularly spinal anæmia, patients can take very large doses of strychnia and get well. I have followed that plan of treatment taught by Brown-Séquard in cases of spinal irritation, giving the first week a thirty-second of a grain, the second week a twenty-fourth of a grain, and so on, and have frequently given a fifteenth and even a twelfth of a grain three times a day. With few exceptions in which the patient showed an idiosyncrasy, no unpleasant effects were produced. My experience has been like that of Dr. Dana, there being extraordinary benefit in both males and females.

With reference to Dr. Jewell's method, I agree with Dr. Gray that it is pretty uncertain and risky practice because of the doubt in diagnosis in many cases. I suggested at the time Dr. Jewell brought his cases before the Association that possibly the reason why he did not get the tetanic symptoms of large doses of strychnia was that his patients had lost the gray matter which when excited produced tetanus. There may have been no central motor apparatus to respond to the large doses of strychnia which he gave. But in cases of transverse myelitis and some cases of central myelitis I have found strychnia illy borne, tetanus being induced by moderate doses, and I have not really dared to carry out Dr. Jewell's treatment thoroughly in any one case. In the treatment of chronic cases, such as those of muscular atrophy and poliomyelitis in the stationary or progressive stage, I have used it moderately and with not over-satisfactory results. I have been in the habit of giving strychnia hypodermically, and have in that way avoided possible errors and have been better able to watch the effect of the drug. In a recent case in which a competent physician had diagnosticated locomotor ataxia, there being certain ataxic symptoms and atrophy in some of the muscular groups, but which I thought was of a functional nature—a neuromimesis,—I gave one fifth of a grain of strychnia once a day without the slightest symptom of poisoning, and with very rapid improvement, as shown by the dynamometer and also by the patient's increased vitality, endurance, and hopefulness. The patient entirely recovered within a few weeks. Indeed I have been led by experience to believe that in many cases the toxic effects of strychnia with small doses might aid us in making a diagnosis betweeen organic and functional diseases of the spinal cord.

The PRESIDENT asked Dr. Seguin whether he regarded the hypodermic administration of strychnia as particularly useful in avoiding its cumulative effects.

Dr. SEGUIN: I have never seen the cumulative effect. In the case of a patient of mine in Pennsylvania, however, the doctor gave a hypodermic injection of strychnia, and was afterward sent for in great haste because of the apparent toxic effects of the drug. But in that case there was possibility of error. The patient was a hypochondriac, suffering from a form of hysteria with imaginary motor trouble. I strongly suspect that the symptoms were more subjective than objective.

Dr. GRAY: In the case of transverse myelitis with secondary contracture the cumulative effects of the drug were produced by its administration in solution. In the other case reported it was given in pill form.

The PRESIDENT then read his address on "Paronymy *versus* Heteronymy as Neuronymic Principles."[1]

Discussion was indefinitely postponed.

Third day, afternoon session.

The President, Dr. B. G. WILDER, in the chair.

The minutes of the previous meeting were read and approved.

Present—Drs. Wilder, Weber, Seguin, Spitzka, Rockwell, Amidon, Jacoby, G. M. Hammond, McNutt, Starr, G. B. Massey, Dana, and Birdsall.

Dr. E. C. SPITZKA then read a paper on "The Relation between the Symptoms and the Lesions of Posterior Spinal Sclerosis."

You are aware that the distribution of the lesions in this disease are not uniform; that in some cases; involvement of the column of Goll is an early, in others a late occurrence, that the direct myelo-cerebellar tract is nearly free in most, and grossly degenerated in a few sufferers from this affection, while special segments of the posterior columns appear to enjoy an almost complete immunity from the destructive process. Let us see whether there is any constant relation between the variation in prominent symptoms, and the variation in the distribution of the lesions in special nerve-tracts.

The cases on which I have based this study are derived from Strümpell, Kahler-Pick, Démange, Hebold, Leyden, Westphal, Babesiu, Takács, Sioli, Wolff, Schultze, Buch, and my own, including one of McBride's, of which I received the specimen from him, for this analysis.

---

[1] See JOURNAL N. AND M. DIS., vol. xii., p. 269, 1885.

The initial lesion of this disease is either in the posterior gray horn or in that part of the column of Burdach traversed by the inner division of the posterior roots as they reach the level of entry into the gray substance. The intensity of the primary lesion is probably in proportion to the rapidity of the development of the disease. It certainly must be regarded as distinct from the true secondary degenerations complicating it. The sclerosis of the column of Goll, the analogous changes in the columns of Clarke, and of the direct myelo-cerebellar tract exhibit a true system-limitation, one which the so-called "root zones" or *bandelettes externes* fail to manifest. I am aware that excellent authority abroad may be cited in favor of the view that the sclerosis of the middle portion of the column of Burdach is a systemic affection. But inasmuch as this part of the posterior column is not made up of fibres of an uninterrupted continuity, it seems difficult to regard it as a system, analogous to the pyramid, the myelo-cerebellar, or the Goll tracts. There is equally good authority for regarding the disease of the middle area of Burdach's column as a slow sclerotic process—whether it be consecutive to the irritation of degenerating nerve tubes as Ranvier claims, or primarily inflammatory as Rumpff asserts, and which for syphilitic cases at least my own preparations sustain. The trabeculæ of connective tissue—dragged in, so to speak, with the posterior nerve-roots—are particularly dense in the lumbar portion of the cord. It may be for this reason that the originating morbid influence concentrates its destructive effects in the so-called posterior *processus reticulares*. At all events such is the fact. It is within this area that certain fasciculi of the posterior roots give off both ascending and descending fasciculi—fasciculi which in transverse lesion of the cord, degenerate for short distances cephalad, and shorter distances caudad. It is reasonable to suppose that this overlapping of ascending and descending root-fibres, associated with the presence of an extra amount of connective tissue, imbedded as this fibre maze is in that part of the cord which is farthest removed from its lymphatic emunctories, affords every temptation to the extension of slow inflammatory trouble. We consequently find that the lesion of the "primary field" remains limited to particular segments of the cord for years, and where the affection was of the "ascending" and not of the "simultaneous" type invades the next segments above in lesser and lesser intensity. Not so, however, with the consecutive sclerosis of the columns of Goll and Foville. If affected intensely at their caudal portions these tracts are found diseased to nearly the same degree and extent for their entire or nearly their entire length, following in this respect the true secondary degenerations. The strongest reasons for regarding the degeneration of the column of Goll in tabes as a true secondary process, due to the cutting off of its nerve supply by the degeneration of the posterior nerve-roots or their gray depôts, are the following: When the lesion of the primary field is limited to the lower lumbar and sacral part of the cord, the degeneration of the column of Goll is limited to its postero-internal border, when the upper lumbar and lower dorsal segment is involved, the column becomes affected nearly in its entity; and when the cervical enlargement is profoundly diseased, an area, lying just laterad of the column of Goll, and which from its cerebral destination may be regarded as its brachial homologue, presents the same morbid condition. In other words, the projection of the sciatic nerve, is in the postero-internal field of Goll's column; that of the crural nerve, more ventrad and laterad in the same column; that of the brachial plexus (and possibly of the dorsal nerves), in the comma-shaped area of Burdach's column, immediately adjoining it.

In all advanced cases of tabes the affection of the column of Goll is in direct proportion to the altitude of lesion in the primary field. Symptomatically it bears an equally direct and constant relation to the degree of motor ataxia. With no case of total degeneration of the column of Goll, do I find motor co-ordination of the local extremities unimpaired; with no case in which there was additional motor ataxia of the arms for any length of time, do I fail to find the comma-shaped area of the column of Burdach uninvolved.

I consider it scarcely necessary to review the opinion of Strümpell, that the lesion of the column of Goll is in relation with the bladder disturbances of tabes.

If the disease of the column of Goll were a primary systemic affection independent of the disease of the root-field, it would be difficult to understand why the likelihood of finding a corresponding degeneration of the direct cerebellar tract increases with the extent to which the former is involved. It is easy however to understand this coincidence, when we bear in mind that both are under the trophic dominion of posterior nerve-roots.

It is in harmony with this fact that static ataxia is found in advanced, or in originally intense, cases of tabes.

Although it be not clearly expressed, the opinion seems to prevail, that the column of Goll degenerates in consequence of a low transverse lesion of the cord. Yet it is not difficult to recognize a difference between the lesion of the tract which corresponds to the column of Goll in the lumbar cord, and the general disease of the posterior column; in many of the plates, the deeper shade of the triangular field, as some have called it, can be distinctly recognized, though the reporters have not accentuated this feature. In the specimen exhibited it is very distinct; it corresponds exactly with the ascending degeneration following compression of the cauda equina, and also with a field which, in a well-preserved fœtal cord submitted to me by Dr. Chauveau, I found to coincide in its myelinic development with the column of Goll, while the field thus designated—with some reservation—by Flechsig, did not so correspond, and remains singularly free from disease in locomotor ataxia. As illustrating the bearing of the lesion of the column of Goll on the motor ataxia, I may refer to two cases related in juxtaposition by Strümpell. As far as the lumbar segment of the cord is concerned, the distribution of the lesion is similar ; in the one without marked ataxia, the triangular field was slightly diseased, and there was no upward extension of the lesion in the column of Goll. In the other with marked ataxia, the triangular field was intensely diseased, and ascending degeneration in the sciatic field of the latter.

In support of the fact, that the comma-shaped area in Burdach's columns is homologous with the fibres of the column of Goll, it is to be advanced, that degeneration of this field bears the same relation to symptoms in the distribution of the ulnar nerve that degeneration of the column of Goll bears to sciatic and crural symptoms. Where the initial pains and subsequent tactile and locomotor disturbance were severe, this field was found affected, and most so on the side where the symptoms had been most intense. This area has no direct connection with the root-fields. Secondly, in primary system-disease of the column of Goll, associated with degeneration of the nucleus of the column of Goll, an irregular encroachment of the column of Burdach was noted. Third, the innermost fibres of the column of Burdach, (those belting the nucleus in the oblongata) have the same relation to the interolivary layer, which the column of Goll has through its provisional nucleus of termination.

While the evidence of high lesion of the cerebral continuation of the column of Goll, and what I regard as its homologue, the comma-shaped area of Burdach, together with the constant association of marked degeneration of these columns with motor ataxia, is strong positive proof of its relation to this symptom, there is equally strong evidence negativing its relation to any other of the prominent symptoms of tabes dorsalis. Thus Babesiu found degeneration of the posterior columns limited to the column of Goll, and the patellar reflex was not destroyed ; the root-fields at the upper lumbar levels were intact. That the columns of Goll have been found profoundly affected without bladder disturbances, has been stated previously, and constitutes a stronger argument against Strümpell's view, than the frequent observation of bladder trouble in spinal diseases with which these columns are entirely free.

Almost the same remarks apply to the relation between static ataxia and the rarer disease of the direct cerebellar tract and the more common affection of the column of Clarke, though the proof is not as ample. The experiments of Woroschiloff and Schiff show that in animals in whom lesion of the pyramid tract is not followed by such profound disturbance as in man, the outer parts of these columns have some relation to co-ordination. The cases of tabes in which either the columns of Clarke, or their cephalic continuation, the tracts of Foville, are degenerated, are advanced cases of the disease, as a rule ; and then, as far as ascertainable, both varieties of ataxia were developed. In Friedreich's form of so-called tabes the myelo-cerebellar tract is more apt to be degenerated, and the ataxia is correspondingly of the static variety as well as of the motor. In one case of profound degeneration of this tract the ataxia is described in terms applicable to cerebellar disturbance. The negative arguments, to which, for reasons to be mentioned, less importance attaches, show that the muscular sense and visceral functions may be disturbed, the patellar reflex absent, the bladder trouble, anæsthesia, and analgesia profound, and yet the columns of Clarke and the tract of Foville be sound ; on the other hand, these tracts may be entirely destroyed without affecting the patellar reflex or the visceral functions. But the valuable case of Senator, in which the cephalic continuation of Foville's tract was destroyed on one side, exhibited a disturbance of the space-sense analogous to that of cerebellar disease. It is the bilateral involvement of this tract which I think renders the cerebellar character of ataxia in advanced tabes so little evident.

The most constant finding, in this analysis, relates to analgesia. Strümpell already noticed that among the divisions of the posterior columns which presented a peculiar immunity to the pathological process, was an area, bordering on the entry line of the posterior roots and the posterior gray horn with one side, and on the periphery of the cord with the other. In a few cases this area was profoundly affected. In the ordinary class there were fulgurating pains, hyperæsthesia, and delayed pain-sense conduction, as in most cases of tabes. In the latter class there was complete analgesia. Now it might be argued against the attributing to this tract of a relation to pain-sense conduction, that naturally in the course of a progressive destroying disease, the phenomena of irritation and hampering of a function will be followed by its abolition. I have, however, here a specimen in which the lesion was remarkably limited : the primary lesion of the root-field ceased in the lower dorsal cord ; the ascending degeneration of the column of Goll was limited to the sciatic field (as I have termed it), and barely identifiable in the cervical region. The " analgesia " area is exceptionally diseased and to a high degree, altogether in disproportion to the disease of the root-fields and the gray substance. The patient, who had been carefully observed by Dr. McBride, had pronounced analgesia of the lower extremities, out of proportion, I think, to the severity of his trouble, generally speaking ; he died of intercurrent disease.

The field of the column of Burdach adjoining the head of the posterior horn in the oblongata is continued in the reticular field of the tegmentum ; lesion here produces abolition of the pain and tactile sense on the opposite side of the body. This much may be affirmed from the cases of tabes reported : in no case in which there was any degree of analgesia were these parts of Burdach's columns healthy, and in no case where they were diseased was the pain-sense preserved.

The symptoms attributed to lesion of the *bandelettes externes* are familiar to all students of Charcot's and Westphal's writings, modifying the location of these bands so as to limit their extension to the periphery. It is now generally conceded that the abolition of the patellar jerk is due to this lesion at the upper lumbar level. Tshirjew has shown that the translation of this reflex occurs in one level of the cord, and a distinct fasciculus may be seen coursing from the innermost root-fibres toward the entero-intermediate cell-group of the anterior horn ; it furnishes a pictorial substratum, if not anatomical proof of the physiological observation. The abolition of the tendon reflexes in the upper extremities ensues with lesion in the corresponding field of the lower part of the cervical enlargement, the exact level not being sufficiently demonstrated by a number of comparable cases. Starr, in his summary, locates the elbow tendon reflex in the level between the fifth and sixth cervical roots, and the wrist tendon reflexes between the sixth and eighth. This is probably correct, as the more remote a motor or sensory periphery of the upper extremity is from the trunk, the lower down will be found its nuclear representative—and that is tantamount to its lower reflex arch.

It was formerly believed, following the clear dictum of Schiff, that retardation of pain-sense conduction was proportionate to the degree of involvement of the gray substance. The profound atrophy of the gray posterior horn in advanced tabes seemed to justify an application of the experimentally obtained fact to tabes dorsalis. Still one objection to attributing delayed pain-sense conduction to the gray substance existed before Erb called attention to the occurrence of this symptom in peripheral disease. Schiff found that pain-sense conduction continued even if very small areas of the gray matter escaped destruction ; in tabes there is never total destruction of the gray substance, large areas escaping. Consequently, the occurrence of analgesia, preceded by delayed pain-sense conduction, would have, in addition to the assumed lesion of the gray, to be supported by a lesion in the white substance—one intense enough either to cut off all the centripetal roots, or the cephalic conductors.

Immediately adjoining the gelatinous head of the posterior horn, there is a column of vertical fibres, which bears an analogous relation to that exhibited by the ascending root of the fifth pair in connection with the gelatinous substance of the *tuber cinereum* of Rolando. Sclerosis of the ascending trigeminus roots has been found in tabes, and in such cases trophic disturbances were marked; thus in Démange's case the teeth dropped out of the upper jaw. In other cases, with and without autopsies, ataxia of the facial muscles was found associated with trophixlesions in the trigeminal distribution ; and it is to be borne in mind that the facial ataxia was attributed to the affection of this root of the fifth pair. If there be a functional analogy between the fibre systems of homologous location to the supposed trophic (?) root of the trigeminus, we may yet find, in the comparative degree of involvement of this tract, some explanation for the variation in trophic symptoms. I am, from the cases examined, only able to say that where there were no trophic disturbances this tract was healthy, but I also found it unaffected in that class of cutaneous lesions which are directly related to the fulminating pains.

In addition to the "analgesia tract," which is but exceptionally involved in tabes, Strümpell found that a deeply situated area of Burdach's column, bordering on the posterior commissure, and which repeats itself in the brachial and lumbar enlargements, presents a remarkable immunity to the disease process.

I am aware that a number of arguments may be advanced against the establishing of a connection between the lesion of the posterior segment of the cord and locomotor ataxia. Westphal, for example, says that lesion of these columns will be found much more frequently when examinations of the cord shall no longer be limited to those cases where disease is suspected from the existence during life of ataxia. He found extensive disease in sufferers from paretic dementia, who did not exhibit the characteristic gait of the tabic patient. But where he could establish the precedence of the organic trouble in the spinal cord, true locomotor ataxia had been present before the insane manifestations exploded. This fact alone seems to prove that the destruction of cortical control is inimical to the development of typical ataxia. Leyden has expressed an opinion in the same direction, when he attributes the lesser manifestness of locomotor ataxia in the female to the inferior cerebral organization. But a more convincing proof in this line is the fact that in proportion as coincident disease cuts off the centrifugal tracts in a posterior sclerosis, the ataxic symptoms become less palpable. This is a frequent observation with the combined forms of sclerosis.

With regard to the very symptom which, at an earlier period was supposed to have a most constant relation to the lesion of the posterior columns, the tactile anæsthesia, the results of this comparative study were least satisfactory. Beyond the confirmation of Strümpell's conclusion, that the columns of Goll, of Clarke, and the direct cerebellar tracts have no connection with tactile transmission of any kind, I am unable to advance propositions.

In the lumbar cord there is a molecular tissue extending in from the entry groove of the posterior roots, and reaching to the gelatinous head of the posterior horn, which embodies spherical nerve cells, and is altered in one case of tabes in my own series. I can find no clear description of this substance anywhere. Although I am unable to attach any special significance to this finding, yet I venture to hope that with the improved methods of histological analysis, devised during the past twelvemonth, attention will be given to this area. It is, from its close relations to the posterior roots, probably of some pathological if not of intrinsic physiological importance.

While it must be admitted that the primary lesion of tabes is competent for each affected level of the root-zones and gray horns, to produce all the characteristic symptoms of tabes referable to that level, yet in the extent and distribution of the consecutive lesions we have valuable gauges of the extent, severity, and duration of the primary disturbance, and we may attribute to the following special disturbances, the ensuing degeneration of the tracts, whose functions are abolished or diminished :

1st.—That of the column of Goll, to the disturbance of muscular sense in the lower extremities.

2d.—That of the comma-shaped internal bands of the column of Burdach, to the corresponding disturbance in the upper extremities.

3d.—That of the triangular area, bordering on the periphery of the cord and the posterior root-entry, to the rarer analgesic development.

4th.—That of the column of Clarke and direct cerebellar (Foville's) tract, to the disturbed space-sense.

The specimens exhibited, Dr. Spitzka stated, were prepared by two methods: one, the ordinary staining of sections as met in ammonio-carmine; the other, of whole segments of the spinal cord. In the latter, the degenerated fasciculi alone imbibed carmine to any appreciable extent; thus the degenerated areas were more distinctly demarcated than by any method in which sections are stained as a whole. Sections prepared by the ordinary methods demonstrated that this demarcation was correct.

Remarks on Dr. Spitzka's Communication.

Dr. E. C. SEGUIN.—Little remains to be said upon a paper which is so comprehensive and analytical as that just read by Dr. Spitzka. I can only express my opinion that it summarizes very well the present state of our knowledge as to the relations which exist between tabes and its symptoms. I would add one series of observations, which are physiological, to the pathological ones, and would ask the author of the paper what possible light they may throw upon the initial lesion of tabes. I refer to the insusceptibility of tabetic patients to the sensory disturbances usually produced by aconitia. Five years ago I made the observation (*vide* Transactions of Meeting of 1881), that patients in the first stage of tabes, and also in the second stage, could take very large doses of aconitia without developing the peripheral subjective sensations which are characteristic of the action of that drug upon the spinal axis. Out of the entire series of cases, perhaps twelve, only one patient manifested any tingling sensation in the extremities during the administration of the aconitia; that patient took enormous doses, as much as six one-hundredths of a grain in the twenty-four hours. Two or three of the patients came near dying from collapse, having the general effects of the drug but none of the characteristic sensory effects. I told them to take the drug until they felt a tingling sensation, which is a safe rule except in tabetic patients. It seems very probable that the tingling produced by aconitia arises from its effects upon the gray matter of the cord, whether by causing a vascular disturbance or a parenchymatous molecular disturbance, I will not venture to say. It has been stated that aconitia acts on the peripheral nerve fibres, but if that were the case, and the nerves were degenerated, I do not see how we could account for the absence of tingling because a very small proportion of the sensory fibres are attacked in early tabes in many cases, as shown by the absence of severe or extensive anæsthesia.

Dr. SPITZKA.—Was there any limitation of this phenomenon?

Dr. SEGUIN.—It was limited to the extremities, as a rule. If, on the other hand, the theory be advanced that the numbness is produced in the gray matter and is then referred to the extremities subjectively, it seems to me difficult to understand the absence of the sensation. Our usual understanding of these subjective sensations is that they take place irrespective of conduction, as shown after amputation, when patients refer sensation to a limb which is absent. It seemed to me from these observations, which are very incomplete and need to be repeated, that there was possibly a physiological proof that there is an early change in the gray matter, perhaps in that part of the gray matter to which Dr. Spitzka has referred as constituting a portion of the posterior horn.

Dr. SPITZKA.—I suppose that in Dr. Seguin's cases the absence of tingling sensation existed only in the extremities, and not, for instance, in the face.

Dr. SEGUIN.—As I remember the cases, there was no tingling or numbness produced at all. Certainly in most of the cases there was no numbness in the lower extremities. In many of the cases there was none in the face.

Dr. SPITZKA.—I agree with Dr. Seguin that from analogy we would infer that the aconitia had not produced sensory symptoms because of change in the gray matter rather than because of a change in the nerve-ends. I do not believe there is a single drug of that class which acts upon the peripheral extremities of the nerves. Perhaps the question could be settled by first giving the aconitia in peripheral nervous trouble, and seeing if transmission would be interfered with. I believe that all these paræsthetic and analogous phenomena have their seat in the gray substance, and for that reason I am inclined to think the initial lesion of tabes is in the gray substance, if not in the posterior roots, because these symptoms predominate in the prodromal stage of tabes.

Dr. SEGUIN.—In certain classes of cases that is true. I have recently seen a patient who had had no pains. He was very certain about that fact. He had had a sense of coldness with crawling in patches, but he denied having had pain in the ordinary sense of the word. His tendon reflexes were absent. He was evidently in the beginning stage of tabes.

The PRESIDENT then read a paper entitled " A Seldom Described Artery (A. termatica), with Suggestions as to the Names of the Principal Encephalic Arteries."

In all the human brains examined in the anatomical laboratory of Cornell University, in which the arteries were preserved and injected, there has been found the small artery which is shown in the preparation exhibited, and represented in my paper on " Encephalie Nomenclature " (*N. Y. Med. Jour.*, March 21, 1885, pp. 325, 327, figs. 1, 3). In that paper it was named *Arteria termatica* (termatic artery), from its location just cephalad of the terma (*lamina terminalis* or *l. cinerea*), which it also supplies with small branches. Its origin will be described presently. It usually divides soon into a right and left portion, which supply respectively the cinerea forming the surface of the triangular area ventrad of the genu and rostrum, and then extend around the genu to the dorsal aspect of the callosum. The ultimate distribution has not yet been determined. The artery merits notice, both because it is *mesal* or *azygous* at its origin, and because, so far as I can ascertain, it has never been named or described, or accurately figured, although a few authors represent some short vessels as given off from the place of origin of the termatic.

In making notes of observations upon this artery, I found somewhat irksome the repetition of even the abbreviations of the dionymic names commonly applied to some of the arteries at the base of the brain, and substituted the following mononyms, which I venture to recommend to this Association. The names *vertebralis* and *basilaris* need no improvement. With all the Latin names, of course, the word *Arteria* is understood, or its abbreviation A., and after the English paronyms the word *artery*. The method of converting dionyms into mononyms is that which was first so aptly applied by Owen to the great veins, *præcava* and *postcava* (permitting the use of adjectives *precaval* or *postcaval*), and adopted by me for the encephalie commissures and some other parts in 1880 (see also "Anatomical Technology," pp. 14, 484, 485).

Common Latin names.	Proposed names.	English paronyms.	Abbreviations.
Vertebralis.	Vertebralis.	Vertebral.	*vrtb.*
Basilaris (*az.*)	Basilaris.	Basilar.	*bslr.*
Cerebellaris anterior.	Præcerebellaris.	Precerebellar.	*prcbl.*
Cerebellaris posterior.	Postcerebellaris.	Postcerebellar.	*pcbl.*
Cerebralis anterior.	Præcerebralis.	Precerebral.	*prcb.*
Cerebralis media.	Medicerebralis.	Medicerebral.	*mcb.*
Cerebralis posterior.	Postcerebralis.	Postcerebral.	*pcb.*
Communicans anterior (*az.*)	Præcommunicans.	Precommunicant.	*prcm.*
Communicans posterior.	Postcommunicans.	Postcommunicant.	*pcm.*
Choroidea anterior.	Præchoroidea.	Prechoroid.	*prchrd.*
Choroidea posterior.	Postchoroidea.	Postchoroid.	*pchrd.*

I have no name to propose for the very awkward dionymic eponym, *Circulus Willisii* (circle or pentagon of Willis), but one is certainly desirable.

In six out of seven brains examined for the termatic artery, it was found to spring only once from a distinct precommunicant artery uniting the precerebrals, as commonly described. In the other cases, as in the preparation exhibited, it arose from the place of junction of the precerebrals, the precommunicant being wholly absent. The precerebrals are joined for only a few millimeters, and then separate again to follow their accustomed course.

Remarks on Dr. Wilder's Communication.

Dr. M. A. STARR, of New York: I would like to state that the anomaly which Dr. Wilder has spoken of, absence of the præcommunicant artery, is one that I have noticed twice within the last year in the demonstration of fourteen brains. If Dr. Wilder has found absence of the artery in five cases out of six it is rather a remarkable fact. The arteries were not injected during my demonstrations, and I did not notice the new artery spoken of.

Dr. WILDER next made a communication "On Two Little-Known Cerebral Fissures, with Suggestions as to Fissural and Gyral Names."

In all adult human brains examined with reference to them in the anatomical laboratory of Cornell University, there have been found, more or less distinctly marked, two fissures which are seldom figured or described, but which probably have considerable morphological significance.

The one indents the dorsi-mesal margin just cephalad of the precentral fissure and paracentral lobule. In the brain exhibited (from an adult mulatto) it is particularly distinct, and is shown in the outline figure in the *N. Y. Med. Jour.*, Feb. 23, 1885, Fig. 42. It seems to have been described by Lussana and Lemoigne (" Fisiologia dei centri nervosi encefalici," Padova, 1871) under the name of *inflesso*; paronymized, in Latin this becomes *fissura inflecta*, and in English the *inflected fissure*. At first glance it strongly suggests the cruciate fissure of the Carnivora, especially when the branches of the precentral, border what resembles the sigmoid gyrus; but such a homology must not be admitted without further investigation. For some of the difficulties involved see the Transactions of this Association for 1883, p. 63.

The other fissure is likewise well developed upon the brain exhibited, which was hardened within the skull by arterial alinjection, and hence escaped the injury and distortion which are sometimes inflicted upon the base, during the ordinary processes of removal and preservation. The fissure appears upon the meso-ventral aspect of the temporal lobe, near its tip, extending caudad for 1–3 cm. from the horizontal portion of the Sylvian, between the neutro-cephalic ends of the hippocampal and collateral fissures. My attention was called to it this spring, and its constancy, and its relation to the Sylvian fissure and the insula led me to homologize it with the postsylvian portion of the fissure which, in the cat, for example, forms the dorso-lateral margin of the tract continued caudad from the olfactory lobe and crus. In man and other primates the great development of the insula and of the true cerebral portion of the hemisphere throws the fissure upon the neutro-mesal aspect, but, there is no essential change in its relations with the parts above named. Later I found that this homology had already been recognized by Owen and Broca in their naming of the fissures of the human brain. Owen's prior name is *basirhinal;* the name employed by me ("Anatomical Technology," p. 496, and elsewhere) is *postrhinal.* If it can be shown to have a constant relation with a constant ental part, the *amygdala*, a name derived therefrom, like *amygdaline*, may be found preferable.

As to the names of the fissures and gyres, on the assumption that, excepting the hypocampal, callossal, rhinal, and part of the Sylvian, all which have definite relations with ental structures, it may be long before the homologies between the primatial and carnivoral parts are satisfactorily determined, I make the following suggestions:

1. That all the indentations of the cerebrum be called *fissures* (abbrev. F.), and this word be restricted thereto.

2. That, excepting a few parts (*insula*, *cuneus*, *præcuneus*, *paracentral lobule*, *uncus*, *subiculum*, *operculum*, etc.) which have received special names, all the interfissural elevations be called *gyres*, Latin *gyri* (abbrev. G.), and that this word be restricted thereto.

3. That, so far as possible, mononymic names for the fissures and gyri be selected from among those which are in use, or formed therefrom by substituting prefixes for words indicating relative position.

4. That in a few cases ambiguous descriptive names be replaced by words indicative of position, relative to other parts having well-established names.

5. That *insula* be preferred to *lobus centralis*, and *central fissure* to *fissure of Rolando.*

6. That the name *Sylvian fissure* be applied only to that portion of the human fissure which is commonly present among other mammals (the "horizontal" or "posterior" branch).

7. That besides the true operculum (between the "posterior" and "anterior" branches of the "Sylvian"), three other opercula be recognized and designated by names indicative of relative position.

In accordance with the above suggestions I have selected or formed the following names for the principal human fissures and gyri. The words here employed are the English paronyms of the corresponding Latin names.

I.—Fissures, mainly or partly mesal.

Callosal, supercallosal, inflected, paracentral, precuneal, occipital, medioccipital, suboccipital, calcarine, postcalcarine, collateral, amygdaline.

II.—Gyres, mainly or partly mesal.

Callosal, paracentral lobule, precuneus, cuneus, postcalcarine, subcalcarine, subcollateral, hippocampal, uncus, subiculum.

III.—Fissures, mainly or partly lateral.

Sylvian, presylvian, subsylvian, basisylvian, olfactory, triradiate, central, precentral, superfrontal, medifrontal, subfrontal, postcentral, parietal, supertemporal, subtemporal, intermediate.

IV.—Gyres, mainly or partly lateral.

Operculum, preoperculum, suboperculum, postoperculum, insula, subfrontal, medifrontal, superfrontal, precentral, postcentral, parietal, marginal, angular, supertemporal, meditemporal, subtemporal, superoccipital, medioccipital, suboccipital.

The paper of Dr. Wharton Sinkler, of Philadelphia, on "Two Cases of Friedreich's Disease," was read by Dr. Massey, of Philadelphia:

The two cases which I shall relate resemble in many respects the so-called Friedreich's disease, but in both is wanting one characteristic feature—namely, the hereditary or congenital history. In all of the cases reported by Friedreich there were two or more instances of the disease in the families to which the patients belonged. Gowers reported five cases of this form of ataxia in a family of nine children.

The cases of Friedreich and Gowers are not, strictly speaking, hereditary, for neither the parents nor any of the ancestors, as far as known, were ataxic. Cases are on record, however, which are hereditary; for instance, those of Carré, reported by Broussi, in which of twelve children seven were ataxic; the mother was ataxic, and her brothers and sisters, eight in number, as well as one cousin, were also affected with ataxia.

As far as I can discover, none of the progenitors of either of the cases which I report are ataxic. The brothers and sisters also appear to be healthy in all respects. I am indebted to the kindness of Dr. Weir Mitchell for the opportunity of reporting the following cases.

Dr. Sinkler then gave the history of two cases of this disease.

These cases, with certain exceptions, are decidedly like locomotor ataxia, but the ordinary form of this disease is very unusual in children. Hammond says it is met with between thirty and forty-five years of age. Eulenburg in 149 cases found but one instance (female) under ten years of age. Erb, out of eighty-six cases of his own, met with three instances between eleven and twenty years.

In a "Critical Digest of the So-Called Hereditary Ataxia, First Described by Friedreich," by Dr. J. A. Ormerod, the writer has collected forty cases of this affection. This author remarks of the disease, that its most striking feature is its tendency to attack many members of the same family. Next, that it attacks in early life. "Friedreich stated that it coincided with commencing or developing puberty, the dates of his cases varying from æt. 13 to æt. 18, but other authorities have observed it still earlier. Thus of Rutimeyer's cases it began above the age of four years (one child indeed had never been able to see straight); and in two of Hammond's cases at a similarly early age." The first symptom is almost invariably ataxia of the legs, then it gradually extends up, affecting the arms, head, and, finally, the speech. Nystagmus also occurs late in the disease. The absence of sensory symptoms is of great interest. In typical cases of locomotor ataxia the pains, as is well known, are a prominent symptom, especially in the early stages; and later on, sensory disturbances—anæsthesia, paræsthesia, etc.—frequently are present. In Friedreich's disease "pain is conspicuous by its absence." "Romberg's symptom"—*i. e.*, inability to stand with the eyes shut and the feet together—is not always present, but the knee phenomenon is always absent; spinal curvature is often present. Ormerod says a combination of lateral and angular curvature is frequent, but that lateral alone is more frequent than angular curvature alone. It is said to be a very early symptom. In my patient, Case 2, there is marked lordosis. Indeed, so marked was this condition that on first seeing the case and observing a very stout condition of the calves of the legs in connection with the spinal curvature I suspected it might be a case of pseudo-hypertrophic paralysis.

Paralysis and atrophy come on later in the disease. Many of the cases recorded were under observation for a great number of years. One of Friedreich's was seen by him for thirty-one, and another for twenty-five years. As the disease progresses, the patient becomes utterly helpless from the degree of paralysis.

Post-mortem examinations were made in seven of the cases collected by Ormerod. The following is a résumé of the changes found:

"In several cases the cord and medulla were small in size, and this is certainly a fact of interest when we consider the hereditary character of the disease, for a cord congenitally imperfect may be the more prone to degeneration, and to degenerate at an early age. Advanced posterior spinal sclerosis was found in every post-mortem. The changes in the medulla were slight and apparently secondary to the spinal. So in Friedreich's opinion were those which he found in the nerve-trunks; while in the cerebrum and cerebellum no trace of chronic disease was found."

In conclusion we would recall the difference in the two different forms of the disease. First, the typical variety begins with lightning pains, disorders of micturition, transient paralysis (especially oculo-motor), absence of pupillary reflex—Argyll-Robertson symptom; visceral crisis; trophic disturbances in the joints and bones. All of these are absent in the form described by Friedreich, and in this latter variety the spread of the affection to the arms is more rapid; the spinal curvature, the disturbance of speech, and the nystagmus are peculiar to it. The early age at which it occurs and the family predisposition to the disease make up the other points of difference.

On the other hand, in both types there are the ataxia and incoördination beginning in the lower extremities, the absence of tendon reflexes "the Romberg symptom," and some of the eye lesions.

Remarks on Dr. Sinkler's Communication.

Dr. SPITZA asked whether there was an autopsy in the two cases reported.

Dr. MASSEY replied in the negative.

Dr. SEGUIN: I have recently published five cases of Friedreich's disease in the *New York Medical Record.* It would perhaps be superfluous to mention the symptoms in those cases except in the briefest way. They presented ataxia of the legs and arms; the Romberg symptom was present; the tendon reflex was absent. There was the progressive characteristic development in early youth, before puberty; in most of the cases decidedly so; in some before the patients had reached four or five years of age. There were three cases in each of the two families. In one of the families one patient had died before I had an opportunity to see her. The point which I think has not been sufficiently treated of by Dr. Sinkler is the distinct paresis and the tendency to contracture which is present in many of the cases. In my own cases the paresis was marked. In a case which I reëxamined about two weeks ago there was pes equino-varus with tendency to contracture of the calf and abnormal tension of the tendo Achilles. The foot could not be brought into normal flexion upon the tibia.

There was increased reflex from the soles, while the patellar reflex was absent. I have been very much impressed in the study of these cases with the absence of pain. It seems to be a rule almost without exception that these subjects have no pain, no anæsthesia. In the celebrated Case 6 of Friedreich, the patient having had the disease thirty-one years, there was no anæsthesia, yet the posterior columns were degenerated as a whole. It seems strange that there should be extensive degeneration of the posterior columns in their different parts and of other portions of the cord without any corresponding irritation or anæsthesia. In the case of the family reported by Dr. Smith before the Massachusetts State Medical Society last week, it was thought that the father had a form of ataxia, although it came on later in life, and there is some doubt as to the real nature of the affection. But the four children certainly had the disease known as Friedreich's disease. Dr. Smith presented one of the children before the Society, and she showed marked paresis and ataxia. There was also a tendency to contracture of the foot. One of the patients died last winter, and the cord after hardening was prepared by Dr. Putnam. I am able to show two sections to the Association, sent on by our Fellow, Dr. Putnam. In these one can see very complete degeneration of the posterior columns, of the cerebellar tract, and of the pyramidal tract. The microscopical examination shows that the pyramidal tract is considerably involved.

With reference to the differential diagnosis of this affection, I think there can be very little difficulty in discriminating between it and disseminated sclerosis. The symptomatology is very typical, whereas in disseminated sclerosis it varies greatly with each case. We have much more decided tremor and quasi-rhythmical tremor in the latter disease. In ataxia the disorder in movement is not a true tremor, but a want of harmony of action between the antagonistic muscles. There was no oscillation of the eyeball, in my cases.

With reference to treatment, I have given two of the patients prolonged treatment with nitrate of silver, iodide of potassium, and galvanism, without the slightest influence on the disease. The cases progressed, as did the other cases which had no treatment.

I will add one word more with reference to the walk in the cases which I saw. There was ataxia of the lower limbs, but it was a sort of imperfect or irregular ataxic movement, but it was not the typical outward projection and then the forcible heel-stamp of tabetic patients. That fact may be of some value in studying the relation between the lesions and thc symptoms.

Dr. R. W. AMIDON.—I think I have under observation two examples of this disease. The one is a girl of eleven years whom I saw last week in consultation. As well as I can recall her history, she commenced to walk very early; I think at the eighth month she walked with the support of a chair. When nine months old she had very severe whooping-cough. Coming out of the whooping-cough she had entirely lost power of progression. This transient paraplegia disappeared completely within three months, and she remained perfectly well, the mother states, up to the age of six years. At that time she began to complain of feeling weak in the legs; she walked in an incoördinate or ataxic manner, and was disposed the latter part of the day to remain perfectly quiet. At the age of seven she had measles, and recovering from the measles her former condition seemed a little aggravated. I saw her last week, at the age of eleven, and her present condition is as follows: She is a very bright girl, and as far as I know is free from any hysterical element. She is believed to be entirely free from any bad habits. She is of normal size. The limbs are very well developed, but there is a feeling in the skin like that described by Dr. Sinkler, as though the skin were not only very thick but were bound down to the subcutaneous tissue. The muscles can be felt very readily under this thick skin and subcutaneous areolar tissue. In the morning the girl gets up and dresses herself and walks about until noon, when she wilts down and does n't walk any more during that day. Her gait is ataxic and stumbling. There are no ocular, vesicle, or rectal symptoms; no pain. I found the muscular reactions to be normal to both galvanism and faradism. The tendon reflex absent.

The other case, which I have not seen for over a year, is that of a girl of five, which I am sure comes under this category of Friedreich's disease described by Dr. Sinkler as not being hereditary. I will endeavor to bring the patient before the Society to-night.

Dr. M. A. STARR—I have very little to say about the section of Friedreich's case in my possession. The case was one of three which Friedreich reported in *Virchow's Archives*. It was a classical case, and one of a number from which the first description of the disease was made. The changes which have occurred in that cord are limited to the posterior columns, to the pyramidal tracts in the lateral columns, and to the direct cerebellar columns of the lateral columns, so that it is really a widespread system of lesions involving three tracts. The cord was also very much deformed in that case; the retraction of the posterior surface of the cord inward making the apparent distance from the posterior commissure to the periphery of the cord very much less than usual. The case was one of three in a family, there being two sisters and one brother. I believe Friedreich regarded it as a case of congenital deformity of the cord, and it is stated that no lesions were found in the brain or cerebellum, which would rather tend to refute the statement attributed to Dr. Hammond, that some lesion of the cerebellum or brain is the primary condition.

Dr. AMIDON.—I would add to my case, that of the girl aged eleven years, that she had a talipes equino-varus, and double tenotomy was performed at the Hospital for the Ruptured and Crippled about two years ago. The tendon Achillis was cut, which, to a certain extent, corrected the deformity, but not entirely.

Dr. JACOBY.—Was hereditary syphilis excluded in Dr. Sinkler's cases?

Dr. MASSEY.—I think syphilis was entirely excluded.

Dr. JACOBY.—Three cases were reported by Hirschberg at the clinic at Berlin, which I think throw some light on the etiology of this affection. Perhaps there were four cases. They came to the eye clinic, and were found to have atrophy of the optic nerve, and also tabes. It struck me in listening to this paper by Dr. Sinkler that his might be cases of tabes in children. They certainly do not strike me as being typical cases of Friedreich's disease as he describes it.

Dr. GEORGE W. JACOBY, of New York, presented an *absolute galvanometer* and a *rheostat*, both of which have been devised by his friend, Dr. J. Rudisch, of New York; a description of them has not yet been published. The principle made use of in this galvanometer is that first employed by Hirschmann, of employing a pair of astatic needles in a vertical instrument. By this means the magnetic influence of the earth is neutralized, and the instrument may be placed and used in any position of the compass. The manner of construction is as follows: Upon two flat spools, made from rectangular copper tube, is wound a sufficient quantity of No. 32 copper wire to make a resistance of eighteen ohms in each spool, each spool being wound separately, the two together having a resistance of thirty-six ohms. The spools, after having been wound, are fastened to a small ebonite base, and stand with their flat sides against each other. The flat side of either spool may be the front of the instrument. Directly over the top and centre of these spools, at right angles with their flat sides, is a small brass shaft of about one and three quarter inches in length, each end of which is capped by a fine steel point, which in turn is received into an agate provided with a conical polished depression. These agates are set into two adjusting screws, which are supported by two posts of such a height as to allow the shaft to turn freely in its bearings. Upon this needle-shaft, and directly over the opening in the spools, are fastened two flat magnets of thin sheet steel. These magnets are each one inch long and a quarter of an inch wide. Directly under these magnets, at right angles to them, and attached to the shaft by means of a forked end, is a small brass arm. This arm hangs down into the interior of the spools for a distance of half an inch, and upon it and parallel to the magnet are fastened eight other needles, each of which is about $\frac{1}{2}$ mm. in diameter. These needles are of various lengths, from $\frac{15}{16}$ of an inch to $\frac{1}{4}$ of an inch, each needle forming a proportionate segment of a circle whose diameter is the sheet-steel magnet, at one end of the needle-shaft, to which the two systems of needles are attached, and very near its supporting agate is fastened a fine aluminium pointer, which extends vertically upward, and swings in front of the scale. This scale is supported by two small pillars, fastened into the top of that spool which is the front of the instrument. The instrument is rendered practically astatic by placing the north and south poles of the systems of needles in opposite directions to each other, the systems being treated the same as an astatic pair of needles.

The *rheostat* almost explains itself. Upon a piece of ground glass is spread a quantity of powdered graphite. Over this glass plate a movable piece of graphite is fastened. This piece forms one of the rheophores, the metal post at one end of the glass plate forming another. The graphite is being continually supplied as the pencil is moved. The advantages over the ordinary water rheostat are manifest. No evaporation, no breakage, and no polarization. The resistance of this rheostat, measured a few days ago, was 30,000 ohms. Of course this resistance is always varying, according to the increase or diminution of graphite. If a very large resistance is desired, it may be obtained by rubbing off some of the graphite with the finger, so that only a slight film remains. That we do not always know the exact amount of resistance interposed is no objection, because we are supposed to take the indication of the galvanometer needle as a guide. Thus, by means of increasing or decreasing the resistance of the rheostat, we can easily increase or decrease the deflection of the needle to the desired point.

Remarks on Dr. Jacoby's Communication.

Dr. C. A. DANA.—It seems to me the galvanometer presented by Dr. Jacoby has a scale too small for indicating the strength of current administered through large electrodes. I should think it could be made to give more than twenty-five milliampères.

Dr. M. A. STARR.—In looking at this galvanometer it does not seem to me to offer advantages over a similar instrument recently constructed by Mr. Barrett, of this city. Dr. Rudisch's instrument is evidently very carefully made, and is, as I know from conversation with the maker, a very expensive one. The number of milliampères indicated on Mr. Barrett's instrument is larger. An attachment of a piece of mica aids in correcting the tendency to vibration. With regard to the point made by Dr. Dana, I know it is the habit here to use large electrodes, but in Germany it is found that twenty-five milliampères represent as strong a current as most persons will submit to. I find that in using Erb's electrodes, if a current of twenty or more milliampères be used, the patient experiences great discomfort, and there is danger, if the current be continued six or eight minutes, of producing eschars which will interfere with the next application. Mr. Barrett originally made his instrument to register fifty milliampères, but I have reduced it to twenty in the instrument I have, because I seldom find it necessary to go above that point with the electrodes of Erb. Giving electricity without any dosage at all is certainly very reprehensible.

Dr. AMIDON.—Am I wrong in the impression that the graduation on the tangent is the same at the extremities as at the middle?

Dr. JACOBY.—The equality of the divisions is only an apparent one, for we have here only a small arc.

Dr. AMIDON.—I cannot see how the astatic needle can record the strength of the current accurately without there be a difference in the length of the degrees marked on this arc. The farther the needle deviates from the central figure, the greater the amount of electricity required to make it move an equal distance. I should think, therefore, that the degrees should be made progressively smaller.

Dr. DANA said that the doubt expressed by Dr. Amidon, with regard to the accuracy of the measurement by these instruments, did not apply practically. He had examined Mr. Barrett's galvanometer, and the same objection had been raised by different gentlemen, but a comparison of his instrument, with those of the central offices of telegraph companies, where it was very necessary to avoid error, showed that the measurements were correct.

The PRESIDENT thought it would be desirable to have some conclusion reached with regard to the accuracy of electrical measurement by instruments of this kind.

Dr. W. R. BIRDSALL had had some experience with galvanometers, and had found that fifty milliampères were not of as great advantage on the ordinary working battery as twenty-five or thirty. With Erb's electrodes twenty milliampères indicated a very strong current; that ordinarily used was from five to fifteen. Mr. Barrett had explained to him the graduation of his instrument, which was done upon the correct plan of graduating according to the tangent system.

A series of brain sections preserved in glycerine, by Dr. H. D. Schmidt, of New Orleans, was presented by Dr. Starr.

Third day, evening session.

The Association was called to order by the President, Dr. B. G. WILDER.

Present—Drs. Wilder, Weber, Seguin, Spitzka, Rockwell, Amidon, Jacoby, G. M. Hammond, McNutt, Starr, G. B. Massey, Dana, Gray, and Bauduy.

The first communication was entitled "Demonstration of a Brain Monstrosity," by Dr. E. C. SPITZKA.

Dr. SPITZKA submitted for inspection the cerebellum and peduncular system of a child which had been affected with a number of peripheral deformities, such as macrodactylism and defective joint and muscle development. The case had been described by Dr. A. Jacobi as one of "congenital lipomatosis." Dr. Boldt, who was the attending physician, and to whom the speaker was indebted for the brain, had informed him that in several details, such as the alleged absence of the patella, the first reports published were imperfect. The cerebral hemispheres were unsymmetrical, and the gyri atypical, so that on one side the sulcus of Rolando could not be identified in any thing like its normal relations. But the most interesting and novel feature was the heterotopia of the cerebellum, shown in the specimen exhibited. This ganglion was in the first place highly asymmetrical; the left cerebellar hemisphere not being one half the size of its fellow, the line of the vermis superior running near the junction of the outer and middle thirds of the transverse diameter. On the right side (basilar aspect) the white substance appeared at the surface like a lobulated tumor, and from it, as a centre, the abnormally arranged cerebellar folia radiated in every direction, so that it was simply impossible to identify the normal lobules. The influence of skull pressure as a modifying factor in the development of the folia was, however, prettily shown.

Dr. SPITZKA also made a communication entitled "Gudden's Atrophy Method."

Dr. Spitzka exhibited two sets of sections obtained from the peduncular axis of a cat, which had been killed three months after destruction of the left cerebral hemisphere and left thalamus had been accomplished. One set had been stained in ammonia-carmine, the other in acid-fuchsine. Aside from the well-known atrophy of the pyramid tract and cortex lemniscus, described by v. Monakow and Mayser, and the bilateral atrophy of the nucleus of the third pair (the nerve having been destroyed on the side of the operation), discovered by v. Gudden, the specimens demonstrated that the posterior commissure is not a commissure, but, as Meynert claims, a decussation of thalamus fasciculi, whose caudal course is immediately subjacent to the posterior longitudinal fasciculus. It also showed that the nuclei of the tegmentum were entirely unaffected, an evidence that the tract of which they are a station is probably a centripetal one. The most marked feature was the enormous atrophy of the nucleus of the column of Burdach, on the side opposite the destroyed thalamus. The posterior longitudinal fasciculus was reduced to from one half to one third or a quarter of the normal in the level of the anterior pair of the corpora quadrigemina, the atrophy being most marked in front and decreasing backward, where complete symmetry appeared to be established.

This atrophy the reporter thought was in connection with the atrophy of the corresponding half of the anterior pair.

Remarks on Dr. Spitzka's Communication.

The PRESIDENT asked Dr. Spitzka what was his impression of the recently promulgated theory, originating in Washington, that the porpoise, although ordinarily credited with having no hind limbs, nevertheless has them and the tail represented in a fused condition. He should say from a study of the nervous system of animals which have hind limbs that it harmonizes exactly with the view that there was absence of those limbs in the porpoise.

Dr. SPITZKA thought that the unity of the different sacral and lumbar nerves in one cord in the canal pointed to simplicity of the part supplied by that cord, and indicated that the porpoise had a true tail.

The PRESIDENT remarked that it showed the necessity for taking into account the muscles and bones in determining questions of homology.

Dr. B. G. WILDER then exhibited preparations of the brains of a dog, monkey (*Cercopithecus*), chimpanzee, porpoise, and man, and of an alinjected human hydrencephal,[1] stating the following points : (*a*) that, contrary to what is expressed or implied in most anatomical works, in the human brain (as in those of all other mammals examined with reference thereto), the rima (the part of the "great transverse fissure" along which the proper nervous parietes of the prosocœle are abrogated and the endyma pushed entad before the intruding prosoplex) stops 1–3 cm. short of the tip of the medicornu (on this point see the *N. Y. Med. Jour.*, April 5, 1884, p. 376, fig. 48) ; (*b*) in the human hydrencephal, notwithstanding the pressure which had more than doubled the normal diameters of the porta (orifice of communication between the aula and either "lateral ventricle," commonly called "foramen of Monro"), the dorsal circumscription, by the endyma reflected upon the intruded prosoplex from the opposite surfaces of the thalamus and fornicolumn, was complete and distinct, as in the preparation represented in the *N. Y. Med. Jour.*, March 21, 1885, p. 328, fig. 7; (*c*) that the diacœle (" third ventricle "), instead of being roofed in directly by the overlying velum or fornix, much less by the callosum, has a true roof of its own, namely, the endyma, which may be distinctly traced in the hydrencephal from the mesal aspect of the thalamus over the habena toward the opposite side (see *N. Y. Med. Jour.*, April 26, 1884, p. 460); (*d*) as shown by Dr. Spitzka before the N. Y. Neurological Society in 1879 and 1880 (*N. Y. Med. Record*, June 25, 1879, and Jan. 17, 1880), other mammals besides man have a more or less elevated or gyrated region concealed by the opercula or lips of the Sylvian fissure. In the dog or monkey there is but a single elevation; in the champanzee two; in man four or five; in the porpoise exhibited the region evidently corresponding to the insula covers a greater area than in man, and the gyri are more numerous (13–15), but their elevation was so slight that Dr. Wilder thought they really represented a less extent of cinerea than in man. He hoped to have an opportunity of preparing the brain of a porpoise by arterial alinjection followed by the injection of starch after Pansch's method, so as to determine accurately the form, extent, and constitution of this important region. In this communication he had availed himself of preparations and drawings made at his suggestion by Mr. F. M. Chappell as a part of his thesis for graduation at Cornell University this spring.

---

[1] After using this mononymic equivalent of *hydrocephalous brain*, I encountered the analogous word *hydrencephalocele*.

In conclusion Dr. Wilder stated that unexpected official duties at Ithaca had prevented him from packing up for transportation the easily injured preparation by which he had hoped to illustrate (*a*) the form and extent of the adult aula (mesal portion of the prosocœle, or cephalad portion of the " third ventricle "), and (*b*) the fimbrial revert (the reverted margin of the fimbria or " *corpus fimbriatum* ") and its relation to the prosoplex (*plexus choroideus ventriculi lateralis*).

Remarks on Dr. Wilder's Communication.

Dr. E. C. SPITZKA.—I regret that I did not bring my specimen of the porpoise brain with me. The insula is not as mountainous as in the human brain; nor would I say now that the gyri are deeper, but they are more numerous. I think the aggregated surface of the porpoise insula would be fully as great, if not greater than the human. I may say that there is almost identity of the gyri in this specimen and in the one I possess; they are only a little more flattened. As the gyri run fan-shaped from before backward they become longer and longer. Not counting one subdivision in my specimen, it contains fourteen gyri.

Dr. WILDER.—Am I to understand Dr. Spitzka to say that the insula is elevated in his specimen?

Dr. SPITZKA.—It is not as elevated as in the human brain, but more so than in the porpoise brain presented by Dr. Wilder. I have the insula of an ourang-outang which contains one gyrus more than the insula of the chimpanzee presented. In the brain of the baboon which I have, there is one sulcus.

Dr. WILDER.—It seems to me the insula affords a very interesting field for investigation, and I wonder that more attention has not been given to it, especially in determining homologous parts.

Dr. E. C. SEGUIN, of New York, then read a paper entitled "A Contribution to the Pathology of the Cerebellum."

The case was one of tumor of the cerebellum occurring in A. M. B., thirty-seven years of age, single, without syphilitic history, who first came under Dr. Seguin's observation in consultation with Dr. English, of New Brunswick, N. J., in 1877. The autopsy was made April 24, 1885, by Dr. English and Dr. Elmer, of Trenton, N. J. Dr. Seguin had diagnosticated tumor of the cerebellum involving the superior vermis.

The symptoms were summarized as follows: Beginning with headache and one or more epileptoid or apoplectiform attacks; impaired vision, optic neuritis, and partial atrophy; typical cerebellar titubation; nystagmus; slight difficulty in articulation; no distinct paralysis; no ataxia, no anæsthesia; patellar reflex raised. Death, with cerebral and bulbar symptoms: stupor, convulsions, difficult articulation and deglutition, ptyalism, and polyuria.

Lesion.—Cyst of the cerebellum destroying the larger part of the vermis, penetrating the right lobe and pressing upon the floor of the fourth ventricle. Extensive syphilitic arteritis with varying forms of softening within the brain.

Remarks on Dr. Seguin's Communication.

Dr. SPITZKA.—Had the patient any subjective disturbance of the sense of space when in a condition of rest?

Dr. SEGUIN.—In the sense of vertigo?

Dr. SPITZKA.—Yes.

Dr. SEGUIN.—He had not. The attacks he described were those of impending loss of consciousness, not vertiginous. Dr. Seguin stated further that the cerebellum was not cut at all, as it was to be examined after hardening.

Dr. R. W. AMIDON.—I recently saw a case of cerebellar tumor, in consultation, in a child about six years of age. When I saw the child it was impossible to determine the subjective symptoms because of the age of the patient and stupor produced by bromide of potassium. The mother about a year previously had been relieved of sarcomatous glands of the neck by Dr. Bull. They did not recur. About a year after Dr. Bull removed the sarcomatous glands from the neck of the mother, this child, then about five years old, was taken acutely with symptoms resembling hydrocephalus. There was no fever, no convulsions. The case progressed four weeks before I saw it. The mother stated that about three weeks previously the child had begun to be very irritable, more or less sleepless, it bored the pillow a good deal with the head, was restless at night, all of the prodromal symptoms of tubercular meningitis. There was slight drooping of both eyelids, moderate external strabismus, probably double. Except when under the influence of bromide of potassium, the child was in perpetual motion. The hearing before the attack had been about normal; it was afterward almost entirely abolished. The optic nerve could not be examined, but vision was pretty good. When I saw the child it had rather a hydrocephalic head, and presented symptoms which led me to make a diagnosis of tubercular meningitis in spite of the absence of fever. But after noticing absence of fever for a week or more I very reluctantly gave up my diagnosis, but did not venture another. There was much muscular weakness, the child was unable to hold up its head; the weakness progressed, finally there came on a condition of stupor, with failure of speech, coma, incontinence of urine, death. At the autopsy there was found a sarcomatous tumor involving the superior vermis, occupied by a cyst. The tumor extended down through the cerebellum, apparently involving to a certain extent the floor of the fourth ventricle. The case was specially interesting because of sarcoma developing in both mother and child.

Dr. DANA.—Were there any forced movements in Dr. Seguin's case?

Dr. SEGUIN.—No. When sitting quietly, the man appeared normal, except a peculiar expression of the countenance, which possibly was natural to him.

Dr. GRAY.—Had the patient the symptom of whirling in a circle?

Dr. SEGUIN.—No, not at all.

Dr. GRAY.—When he had these paroxysms was there any tightening of the skin of the scalp? I have seen in two cases of cerebellar trouble, tightening of the skin of the head as on a drum-head.

Dr. SEGUIN.—I did not hear of that in my case.

Dr. SPITZKA.—Was there any thing abnormal in the position maintained by the head?

Dr. SEGUIN.—There were no vibrations during rest, but there was a little inclination forward.

Dr. SPITZKA.—I am reminded of a remarkable case from the point of view of histology as well as of symptomatology—a case of pure lipoma of the inferior peduncle of the cerebellum. It was as if the part of the cerebellum left in Dr. Seguin's case were replaced by the lesion; the lesion, however, was more to the left than to the right side. The patient was a tailor afflicted with a form of insanity. He could work at his trade only by having a support for the head. Because of the fact that he had this psychosis and I saw him rarely I gave but little attention to his symptoms, and hence discovered the tumor only by accident.

The next communication was by Dr. BAUDUY, of St. Louis, " Notes on Hydrochlorate of Cocaine."

A paper by Dr. PHILIP ZENNER entitled " The Psychomotor Centres," a clinical contribution, and a paper by Dr. H. D. SCHMIDT, "A Case of Destructive Lesions of the Tegmentum, Thalami, and other Parts of the Brain," (see JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. XII., p. 294), were read by titles.

The PRESIDENT appointed as Committee on Electrical Dosage, Dr. Geo. W. Jacoby, Chairman; Dr. Amidon and Dr. Birdsall.

On motion, the Society adjourned to meet at about the same time next year, and at a place to be determined by the Council.